

# RESPIRATORY SYSTEM

## APPROACH TO SHORTNESS OF BREATH

Few imp D/D's of acute shortness of breath

- less likely to encounter such patients in exam

### 1. Cardiac Causes

- Acute MI( cardiogenic shock)
- Acute Pulmonary Edema
- Acute Valvular Insufficiency
- Acute Aortic Dissection( a young gentleman diagnosed case of Marfan syndrome once was a long case in Islamabad centre)
- Acute Pericardial Tamponade

### 2. Respiratory Causes

- Pneumothorax
- Acute Pulmonary Embolism
- Inhale Foreign Body (may cause lobar collapse)
- Acute Asthma (previously undiagnosed)
- Acute Exacerbation of COPD(previously undiagnosed)
- ARDS
- Acute Massive Pleural Effusion

### 3 Miscellanies

Metabolic Acidosis like DKA and lactic acidosis etc.

Anaphylaxis

Psychogenic hyperventilation

Important D/D, s of chronic shortness of breath and their description

**Likely to encounter such patients in CPSP exam**

### 1 cardiac Causes

❖ CCF → Hx of SOB, pink frothy sputum, distended neck veins and gen body swelling.

#### ❖ Valvular heart disease

- Rheumatic heart disease
  - HX of monthly intramuscular injection, abnormal movement of any part of body
- Non- Rheumatic heart disease
- Congenital

#### ○ Cardiomyopathies

- Dilated cardiomyopathy

HX of alcohol abuse, chemo (doxorubicin) and radiation exposure etc.

- Restrictive cardiomyopathy

Past /Contact HX of TB (low grade fever weight loss, night time sweating with often blood stain)



sputum

- **Hypertrophic Cardiomyopathy**

HX of sudden cardiac death in family

- **Ischemic heart Disease**

HX of smoking, DM, HTN, lipids disorders, sedentary lifestyle.

- **Infective endocarditis**

HX of IV drug abuse, recent instrumentation of the body & surgery, tooth extraction, high risk sexual behaviour etc.

## 2 RESPIRATORY CAUSES

- **Asthma**

HX of SOB with dry cough that is worse in the late night & early morning e.g. diurnal variation and associated with wheezy chest sounds

- **COPD**

HX of SOB with productive cough that is worse during the winter season on the background of chronic smoking or biomass exposure

- **Pneumonia**

HX of high-grade fever, rigors and chills with rusty sputum

- **Pulmonary TB**

HX of low-grade fever, night sweats, weight loss and productive cough that is sometime stain with blood

- **BRONCHIECTASIS**

HX of copious amount of foul smelling sputum that is worse during the morning and at certain position

- **ILD**

Hx of SOB and dry cough and past Hx of exposure to factory, particles, fumes, farm, dusts, drugs & radiation or known case of connective tissue disorder.

- **PLEURAL EFFUSION**

Hx of collection or drainage of fluid from chest cavity

- **Cor Pulmonale**

Hx of some lung pathology, now presented with lower limbs & abdominal swelling, prominent neck veins and tender right hypochondrium (congestive hepatomegaly)

- **SARCOIDOSIS**

Painful skin lesion on shin

- **CA LUNG**

Hx of weight loss, decrease appetite, change in the quality of voice (hoarseness), difficulty in swallowing and lumps and bumps in the body.

## 3. AUTOIMMUNE DISORDERS

- **Connective tissue disorders and vasculitis**

*A general description*

Hx of oral ulcers, photosensitivity, hair fall, arthralgia or arthritis, (palpable or) petechial skin rash and blood in sputum or urine plus constitutional symptoms.

#### **4 Anemia**

Hx of SOB associated with Change in the complexion, palmar and conjunctival pallor, racing of heartbeats and easy fatigability that is non-fluctuating.

#### **5 CKD**

Hx of decrease urine output, blood in the urine or frothy urine and patient is on renal replacement therapy.

#### **6 CLD**

Hx of blood in the vomitus, black tarry fould smelling stools and abdominal distension +\_ yellow discoloration of sclera.

#### **7 Thyroid Disorder (Hypo/Hyper )**

Hx of associated with heat intolerance, excessive sweating, involuntary movements of hands, loose motions and irritability +\_ swelling in the neck.

Hx of cold intolerance, weight gain, periorbital puffiness, sluggish mental activities, dry rough skin and hair +\_ swelling in the neck

#### **8 Neuromuscular Disorder**

**MG, MND, GBS etc.**

Hx of fluctuating weakness that is more at the end of the day associated with double vision or regurgitation of foods and difficulty in swallowing.

#### **9 Recurrent thromboembolism**

Hx of prolong embolization, recent travel, family Hx of lower limb swelling (DVT due to hypercoagulable state



## APPROACH TO SOB (Due to COPD)

### Presenting complaints

- (1) Worsening of shortness of breath... 7 day or
- (2) Cough

### HOPI (& ODPARAV)

MY patient XYZ normotensive ,normoglycemic ,chronic smoker for 30 years ,having recurrent shortness of breath over the past years '' now presented with worsening of shortness of breath for the last 7 days.

- Old-aged examiners famously known as babas do not like to listen premorbid like diabetic or hypertensive /normotensive, norm glycaemic or any condition in the beginning of HOPI while young examiners do.

Shortness of breath started gradually and is progressive in nature .Initially, the patient experienced shortness of breath on moderate exertion like, on climbing stair; however fort the last one-week patient became short of breath even on walking few steps on the plain ground. This shortness of breath was aggravated by exertion & lying flat on the bed therefore my patient use 2 pillows to prop-up himself. Initially SOB was relived with use of inhaler ,however now for the last 7 days despite using inhaler patient remained short of breath throughout the day and night .it is worth mentioning that his inhaler technique is correct. Shortness of breath is associated with cough with scanty sputum production that is non- purulent having no blood and is more at night. It is also associated with chest tightness and wheezy chest sounds. There is also associated history of seasonal variation of these symptoms in the winter and HX of skin allergies .However there is no associated history of ...

Now exclude few important relevant DDs of shortness of breath (CPSP examiners expect you to rule out important DDs of each disease like COPD here even if you are 200% sure that this is the case of COPD.

- **Bronchial Asthma** → There is No HX of SOB & dry cough with diurnal variation that is associated with wheezy chest sounds & Hx of atopy and family history of asthma
- **Pneumonia** → There is no HX of high-grade fever, rigors and chills with rusty sputum
- **Pulmonary TB** There is no HX of low grade fever , night sweats, weight loss and productive cough that is sometime stain with blood
- **Bronchiectasis** → There is no HX of copious amount of foul smelling sputum that is worse in the morning and expectorate at certain position
- **ILD** → There is no Hx of SOB and dry cough with past Hx of exposure to factory, particles, fumes, farm, dusts, drugs & radiation or known case of connective tissue disorder .
- **Pleural Effusion** → There is no Hx of fluid collection in chest cavity
- **Cor Pulmonale** → There is no Hx of prominent neck veins , tender right hypochondrium and lower limb swelling due to some lung pathology
- **CA- lung** → There is no Hx of blood in sputum that is associated with weight loss, decrease appetite, change in the quality of voice (hoarseness), difficulty in swallowing and lumps



and bumps in the body

- **Lung Abscess** → There is no HX of any chronic immunocompromised condition e.g. IV drug abuse DM etc. that is associated with high grade fever associated with chills and chest pain
- **sarcoidosis** → There is no HX of painful lesions on shin that is associated with dry cough
- **CCF/CHF/Cardiac Asthma** → There is NO Hx of SOB that is associated with pink frothy sputum, distended neck veins and gen body swelling .or
- There is no Hx of SOB associated with disturbed night sleep that is relived with prop-up position
- **Rheumatic heart disease** → There is no HX of monthly intramuscular injection , abnormal movement of any part of the body
- **Ischemic heart Disease** → There is no Hx of SOB associated with left sided chest pain on exertion on background of smoking ,DM ,HTN, lipids disorders , sedentary lifestyle
- **Infective endocarditis** → There is no HX of IV drug abuse ,recent instrumentation of the body & surgery ,tooth extraction ,high risk sexual behaviour
- **Connective tissue disorders and vasculitis** → there is no Hx of oral ulceration, photosensitivity, hair fall, joint pain, skin rash, skin tightening and blood in sputum or urine → **Connective tissue disorders and vasculitis.**)
- **Anemia** → There is no Hx of SOB associated with Change in the complexion, palmar and conjunctival pallor, racing of heart beats and easy fatigability
- **CKD** → There is no history of SOB associated with decrease urine output, blood or frothy urine and gen body swelling
- **CLD** → There is no Hx of blood in the vomitus, black tarry fould smelling stools and abdominal distension
- **Hyperthyroidism** → There is no Hx of associated with heat intolerance, excessive sweating, and involuntary movements of hands, loose motions and irritability ± swelling in the neck
- **Hypothyroidism** → There is no Hx of cold intolerance, weight gain ,periorbital puffiness, sluggish mental activities , dry rough skin and hair ± swelling in the neck
- **Neuromuscular disorder** → There is no Hx of fluctuating weakness that is more at the end of the day associated with double vision or regurgitation of foods and difficulty in swallowing
- **Thromboembolism** → There is no Hx of prolong embolization , recent travel or surgery , family Hx of lower limb swelling
- Exclude only few important relevant DDs in long case to save time otherwise you may not



be able to complete the long case within 25 minutes.

Now apply CARE/RECA of your case

(COPD here)

CARE (Also Called RECA) stand for Aetiology, Risk Factors, Complication and Association

#### Aetiology and Risk Factors

As I mentioned earlier my patient has history of smoking of 30 pack years. He is very well aware of the adverse effects of smoking but he has never attempted to quit smoking. However, there is no history of exposure to biomass, fuel, farm, factory, mines, animals, pets, birds, fumes, dust and gas particles. My patient denied HX of yellow discoloration of sclera and lung disease in the family members (Alpha 1 antitrypsin deficiency). (So in the patient COPD is mainly due to smoking)

Now ask about the major complication of your long case, HX

#### Complications of COPD

1. **Right heart failure / Cor Pulmonale** there is HX (or no Hx as per your case findings) of distended neck veins, abdominal and lower limb swelling and yellow discoloration of sclera (Congestive Hepatopathy).
2. **CO<sub>2</sub> retention/narcosis** there is Hx of early morning headache, altered sensorium and feeling of excessive warmth in the body
3. **Secondary Polycythaemia** There is no HX of redness of the skin, whistling sounds in the ear and itching after hot bath.
4. **Respiratory failure and Cyanosis** Hx of bluish discoloration of mucous membrane/ body and invasive/ non-invasive ventilation during hospital admission
5. **Lung CA** Hx of decrease appetite, change in the quality of voice (hoarseness), difficulty in swallowing and lumps and bumps in the body, muscle weakness and bone pain, constipation and dry mouth.
6. **Pneumothorax** HX sudden severe chest pain and shortness of breath that was relieved with chest drain
7. **Repeated chest infection** HX of purulent sputum with fluid collection in chest cavity
8. **Medication like Steroid side effects** HX weight gain, excessive hair growth, acne, newly diagnosed DM, HTN, and hoarseness of voice e.g. iatrogenic cushingoids.

Now inquire about ITP i.e. relevant investigations, treatment and any procedure (spirometry and bronchoscopy etc) done during the current admission.

Now Ask About RELEVANT

PAM HUGS FOSS TV F.

P → PAST HX

"HX dates back when my patient presented with SOB and went to a particular hospital or doctor etc. and describe briefly how and who diagnosed the disease e.g. what specific investigations were done like pulmonary function tests including spirometry and what was the result of those tests. Ask about at least 2-3 most recent hospital admission. Describe each in detail including investigation, treatment including invasive and non-invasive interventions. Any other significant pre-morbid condition and inquire about it according to your time management.

Remember relevant past history is the part of the history of present illness.



- **P** → personal HX ;like weight appetite sleep and bowel and bladder
- **A**→ Allergies HX ;ask about any allergies'
- **M**→ Medications HX ;Ask about medication frequency and dosage and its adverse effects
- **H**→ Hospitalization HX; ask how many times, reason and management including any complications.
- **U & G**→ (URO-GYNE HX)
- **S**→ Surgical HX; Bullectomy and lung volume reduction surgery etc
- **F**→ Family HX; ask about family HX of COPD (alpha 1 antitrypsin deficiency)
- **O** →Occupation (occupational exposure to coal dust ,silica ,cadmium)
- **S** → Sexual HX
- **S** → Socioeconomic HX (ask relevant socioeconomic Hx like factors that may prone him to COPD (or its exacerbation) and ask who take care of him including physical and financial support).
- **T** →Travel/Transfusion HX
- **V**→ Vaccination HX (Covid-19 , H influenza and pneumococcal)
- **F** →Functional status (include physical and mental impact of the disease).

### **Examination of COPD**

- Start your examination after 8-10 minutes and take remaining history simultaneously.
  1. **Vitals:** First of all, measure BP at the right arm and put the thermometer in left axilla to record temperature.
  2. **GPE:** look for ;

Nicotine stain, flapping tremors, pepper thin skin , bruises , wasting of small muscle of the hands, palmar erythema, bounding pulse ,evidence of proximal myopathy, lymph nodes, JVP normal or raised , oral thrush ,anemia and jaundice, thyroid and pedal edema.

### **3. Systemic Examination**

Request the patient to sit and expose him adequately. Now examine the back thoroughly according to the standard practice then examine front of the chest along with trachea, tracheal tug and measure cricosternal distance.

Next do CVS examination particularly look for signs of pulmonary hypertensions and right heart failure including comment on JVP.

During abdominal examinations, look for ascites, hepatosplenomegaly and purpura.

In neurological examination look for drowsiness confusion and syncope (type II resp failure) and do quick examination of motor and sensory system of upper and lower limbs including cranial nerves and Gait.

At the end of the examinations ask permission to check orthostatic hypotension (because CA LUNG may metastasis to adrenals) and do fundoscopy for papilledema that may be due to either metastasis or CO2 narcosis.



## Presentation of the examination

### On examination

- An old man who is thin, lean and emaciated sitting on a couch and remained co-operative throughout examination. He was in obvious respiratory distress.
- There was nicotine staining of 2<sup>nd</sup> and 3<sup>rd</sup> finger of the right hand. There was flapping tremor, bounding pulse that was in the range of 80-90 beats/minute and regular his BP was---- and temperature recorded--- C. Hands were warm. However, there was no clubbing, palmar erythema and cyanosis.
- There was redness of complexion, oral candidiasis and poor oral hygiene.
- JVP is normal/ raised and there was no lymphadenopathy.
- On examination of the back: His Chest was barrel in shape and moving more in the vertical plane with predominantly abdomino-thoracic pattern of respiration having a respiratory rate of 25 breaths per minute. He was using accessory muscle of respiration with intercostal, supraclavicular and infraclavicular recession. There was suprasternal and supraclavicular space excavation and there was pursing of lips. However, there was no gross chest deformity, prominent veins, scar marks and visible pulsation.
- Trachea was central with positive tracheal tug and cricosternal distance was decreased (less than 3-fingers). Apex beat was not palpable. Chest movements were bilaterally reduced and chest expansion was less than 3 cm. Vocal fremitus was reduced on both side.
- Percussion note was hyper resonant in both lung fields and there was loss of cardiac and liver dullness.
- The intensity of breath sounds was decreased with normal vesicular breathing with prolonged expiration. Expiratory rhonchi were scattered throughout the chest, and vocal resonance is also reduced
- Additionally, there was a left parasternal heave, and the first and second heart sounds were audible. The pulmonic component of the second heart sound was louder in pulmonic area (mention murmur if present).
- The liver was palpable, but the liver span was normal, and there was no evidence of free fluid in the abdominal cavity. There was/ no sacral or pedal edema.
- Motor, sensory systems and cranial nerves were unremarkable.
- GAIT examination was remarkable for proximal myopathy (Gower sign was positive).
- Fundoscopy was unremarkable.

So based on my history and examination, my clinical diagnosis (or differential diagnosis) is

1. COPD ( more specifically emphysema or chronic bronchitis )
2. Chronic persistent asthma
3. Bronchiolitis obliterans ( but there will be no HX of smoking and inspiratory ronchi on chest examination )

You must defend your first diagnosis, 2<sup>nd</sup> and 3<sup>rd</sup> differential diagnosis.



## VIVA OF COPD

### What is COPD?

#### GOLD 2023 Definition of COPD

"COPD is a heterogeneous lung condition characterized by chronic respiratory symptoms (dyspnoea, sputum production, exacerbations) due to abnormalities of the airways (bronchitis and bronchiolitis) and /or alveoli (emphysema) that causes persistent, often progressive, air flow obstruction."

#### How would you investigate this patient?

- CBC → raised TLC , anemia & secondary polycythaemia
- ESR and CRP marker of infection
- S/E esp potassium
- ECG for RVH , MAT and AF
- X-ray chest PA view

No reliable radiographic signs that correlate with the severity of air flow limitation, however, it is essential to exclude alternative diagnosis like cardiac failure, lung cancer. Following feature may be visible on CXR in COPD patient.

- Hyper inflated lung field
- Low flat diaphragm
- Hyper lucent lung fields
- Long tubular heart shadow
- Bullae
- ABGs for respiratory failure( inability to keep oxygen and/or CO<sub>2</sub> levels within normal limit is respiratory failure)
  - Type 2 failure in chronic bronchitis
  - Type 1 in emphysema
- ECG RVH and pulmonary HTN
- 2D ECHO for cor Pulmonale
- Sputum for microscopy and culture & sensitivity.

#### Specific Tests

- **Pulmonary function test** including objective evidence of airflow obstruction by spirometry (PUNK VALA TEST HWA THA?) Post bronchodilator FEV<sub>1</sub>/FVC < 70 % is diagnostic. Other findings include increased total lung capacity, increased functional residual capacity and residual volume. Decreased vital capacity and TLCO (CO transfer factor).
- If young patient with predominantly basal emphysema than alpha one anti-trypsin level.
- **Helium dilution technique** for measurement of lung volumes that in turn provide an assessment for hyperinflation, however in patients with severe COPD and those having large bullae then body plethysmography is preferred.



- HRCT for characterization and quantification of emphysema and bullae

#### What Are the Risk of COPD?

Environmental factors	Host Factors
<ul style="list-style-type: none"> <li>• Indoor air pollution in developing world</li> <li>• Occupational exposure to silica, coal dust and cadmium</li> <li>• Low birth weight, may reduce maximally attained lung function in young adult life</li> <li>• Recurrent infection, accelerate decline in FEV1</li> <li>• Low socioeconomic status</li> <li>• Cannabis smoking</li> </ul>	<ul style="list-style-type: none"> <li>• Genetic factors alpha one anti-trypsin deficiency</li> <li>• Airway hyper- reactivity.</li> </ul>

#### How will you assess severity of COPD?

Traditionally severity has been defined based on EFV % predicted. However, clinically relevant assessment of severity is based on impact of COPD on patients in terms of symptoms and limitation in activity and frequent and significant exacerbations.

#### Spirometric classification of COPD severity based on post -bronchodilator FEV1

Global initiative for obstructive lung disease (GOLD)

Severity of airflow obstruction Post -bronchodilator FEV1/FVC <0.7

FEV <sub>1</sub> /FVC	FEV <sub>1</sub> % Predicted	Stage	NICE Clinical guidelines 101 (2010)
<0.7	≥80%	Stage- I Mild	Stage I- mild
<0.7	50-79%	Stage-II moderate	Stage II- moderate
<0.7	30-49%	Stage-III severe	Stage III- severe
<0.7	<30%	Stage-IV very severe	Stage IV- very severe

#### How would you manage this patient?

1. Smoking cessation

#### What are the pharmacotherapies and pharmacological products for smoking cessation?

- Nicotine replacement therapy (NRT) like nicotine gums, inhaler, nasal spray, transdermal patch, sublingual tablets.
- Contraindications of NRT include recent MI or stroke. Start two weeks after the event.
- Bupropion, nortriptyline and clonidine but the last drug is limited by its adverse effects.
- **How to calculate packs years** = (no of cigarette /20) \* no of years



2. Drugs according to the severity
- According to GOLD 2023 ABE TOOL (old was ABCD TOOL).
  - A patient may fall into one of 3 categories. I.e. A B E based on assessment of symptoms /risk of exacerbation, mMRC and CAT score and would be treated accordingly.

#### MMRC Dyspnoea Scale

Grade	Degree of breathlessness related to activities
0	No breathlessness, except with strenuous exercise
1	Breathlessness when hurrying on the level or walking up a slight hill
2	Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100 m or after a few minutes on level ground
4	Too breathless to leave the house, or breathless when dressing or undressing

#### What is CAT?

COPD assessment test called CAT is an 8-item questionnaire that assesses health status in COPD patient. The score range from 0 to 40.

❖ Do not memorized CAT score but only MMRC dyspnoea scale

#### Category A

Patients having mMRC 0-1 and CAT <10.

#### Category B

MMRC  $\geq 2$  and CAT  $\geq 10$ .

While both categories may have 0 or 1 moderate exacerbation not leading to hospitalization

#### Category E

$\geq 2$  moderate exacerbation  $\geq 1$  leading to hospitalization.

#### Initial Pharmacological Treatment Based On ABE Tool

##### Group A

A bronchodilator (SABA (Salbo) or SAMA (tiova))

##### Group B

LABA+LAMA\*

##### Group E

LABA+LAMA\* (TIOVAIR F)

Consider LABA+LAMA+ICS\* if blood eosinophil's is 300 or more.

If patients treated with LABA+LAMA+ICS still have exacerbation then add Roflumilast.

- Roflumilast (PDE-4 inhibitor) has been shown to reduced exacerbation frequency in patients who have moderate or severe (FEV1 <50% of predicted) COPD and chronic



bronchitis with frequent exacerbation and who are taking LABA/inhale corticosteroids with or without a LAMA.

- Add a macrolide azithromycin particularly in those who are ex-smokers.

#### **Vaccination in COPD**

- Influenza
- Covid-19
- One dose of PCV20 or PCV 15 followed by 23- valent pneumococcal polysaccharide vaccine(PPSV23) as per CDC recommend Tdap vaccination against pertussis( whooping cough) for COPDier that were not vaccinated in adolescent and zoster vaccine for those who are above 50 years .

#### **What are the therapeutic interventions that reduce mortality in COPD?**

- **Pharmacological therapy**
  - According to IMPACT and ETHOS clinical trials fixed -dose inhaled triple combinations (LABA+LAMA+ICS) reduce all -cause mortality compare to dual long-acting bronchodilation therapy.
- **Non -pharmacological therapy**
  - Smoking cessation
  - Pulmonary rehabilitation(PR)
  - Long term oxygen therapy(LTOT)
  - Non-invasive positive pressure ventilation(NPPV)
  - Lung transplantation and lung volume reduction surgery (LVRS).

#### **What is pulmonary rehabilitation (PR)? Moreover, what are its advantages?**

It is defined as , a comprehensive intervention based on thorough patient assessment followed by patient-tailored therapies that include ,but not limited to, exercise training ,education, self-management intervention aiming at behaviour change, designed to improve the physical and psychological condition of people with chronic respiratory disease and to prompt the long -term adherence to health -enhancing behaviours.

##### **Advantages**

- PR Improves dyspnoea, health status and exercise tolerance in stable patients.
- PR reduces hospitalization among patients who have had a recent exacerbation e.g. 4 or less than 4 weeks from prior hospitalization
- PR leads to a reduction in symptoms of anxiety and depression.

#### **What do you know about Tele-rehabilitation?**

It's new model of rehabilitation that is still evolving and best practices are not yet established.

#### **What are the indications of LTOT?**

1.  $\text{PaO}_2 < 55\text{mm Hg}$  ( 7.3kPa) or  $\text{SaO}_2 < 88\%$  or
2.  $\text{PaO}_2 > 55$  but  $< 60\text{ mmHg}$  ( $> 7.3\text{ kPa}$  but  $< 8\text{ kPa}$ ) with one of the following
  - right heart failure /Cor Pulmonale ( peripheral edema)
  - Secondary polycythaemia



- Nocturnal hypoxemia:  
2-4 L/min oxygen at least 15 hours/day to keep  $\text{SaO}_2 \geq 90\%$  without unacceptable rise in  $\text{PaCO}_2$

#### What are the benefits of LTOT?

- Improve secondary polycythaemia
- Decrease salt & water retention
- Decrease cardiac arrhythmias
- Decrease sympathetic outflow
- Improved quality of sleep at night due to reduced hypoxia induced sleep arousal.

#### How would you assess COPD patient for LTOT?

By measuring ABGs on two occasions at least 3 weeks apart in stable patient on optimum management and at least 4 weeks after exacerbation.

Active smoking is contraindication to LTOT.

#### Management of Exacerbations

Exacerbation is defined as an event characterized by dyspnoea and /or cough and sputum that worsen over <14 days which may be accompanied by tachypnea and /or tachycardia and is often associated with increased local and systemic inflammation caused by infection, pollution or other insult to the air-way.

##### DDs of acute exacerbation of COPD

- Pneumonia
- Heart failure, myocardial infarction and cardiac arrhythmias
- Pneumothorax and pleural effusion
- Pulmonary embolism.

#### How would you manage an exacerbation of COPD?

##### Summary for management of acute exacerbation as per GOLD 2023 guidelines

- Administer control oxygen therapy when required.
- Short acting inhaled beta2-agonists, with or without short-acting anticholinergic, is recommended as the initial bronchodilators to treat an exacerbation.
- Maintenance therapy with long-acting bronchodilators should be initiated as soon as possible.
- Inhaled corticosteroid should be added to those patient having frequent exacerbation or raised peripheral eosinophil levels
- In severe exacerbation, systemic corticosteroids can improve lung function (FEV1), oxygenation and shorten recovery time including hospital stay.
  - Duration of therapy should last no longer than 5 days.
- Antibiotic, when indicated, can shorten recovery time, reduce risk of early relapse, treatment failure and hospital stay.
- Non-invasive mechanical ventilation should be first mode of ventilation used in COPD patients with acute respiratory failure who have no absolute contraindication because it improves gas exchange, reduce work of breathing and need for intubation, decreases hospital



stay and improve survival.

- Exacerbation recovery times vary among patient taking 4-6 weeks.
- monitors fluid balance
- Consider subcutaneous low molecular heparin for thromboembolism prophylaxis
- Identify and treat associated conditions like heart failure, arrhythmias etc
- Determine the aetiology for exacerbation e.g. sputum culture, viral testing, CXR and others.

#### Pathogen causing exacerbations

1. Haemophilus influenzae (most common)
2. Streptococcus pneumoniae
3. Moraxella catarrhalis
4. Respiratory viruses like human rhinovirus being the most important one.

#### What is the prognosis of COPD?

Prognosis of COPD is predicted by BODE index:

Variables	BODE index			
	1	2	3	4
FEV <sub>1</sub>	≥65	50-64	36-69	≤35
Distance walked In 6 mins(m)	≥350	250-349	160-249	≤149
MRC Dyspnea scale	0-1	2	3	4
Body mass index	>21	≤21		
A patient with a BODE score of 0-2 has a mortality rate of around 10% at 52 months, whereas a patient with a BODE score of 7-10 has a mortality rate of 80% at 52 months				

#### What are the poor prognostic factors of COPD?

- Advancing age and fall in FEV1 (most important one)
- Weight loss
- Pulmonary hypertension

#### How would you classify the severity of COPD exacerbations?

It is classified into 3 types

##### MILD

- Dyspnoea VAS\* < 5
- RR < 24 breaths/min
- HR < 95 bpm



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- Resting SaO<sub>2</sub> 92% ambient air
- CRP < 10mg/L

#### **MODERATE 3 out of 5 points**

- Dyspnoea VAS  $\geq 5$
- RR > 24 breaths/min
- HR > 95 bpm
- Resting SaO<sub>2</sub> < 92% ambient air
- CRP  $\geq 10$ mg/L
- ABGs may show hypoxemia (PaO<sub>2</sub>  $\leq 60$  mmHg) and /or hypercapnia (PaCO<sub>2</sub> > 45mmHg) but not acidosis.

#### **Severe**

Moderate COPD but ABGS shows hypercapnia and acidosis (PaCO<sub>2</sub> > 45mmHg and PH < 7.35) is severe exacerbation of COPD.

- VAS\* Visual analog dyspnoea scale.

#### **What are the indications for Hospitalization?**

- Severe symptoms such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, confusion, drowsiness.
- Acute respiratory failure
- Onset of new physical signs e.g. cyanosis, peripheral edema
- Failure of an exacerbation to respond to initial medical management
- Presence of serious comorbidities eg heart failure etc.
- Insufficient home support.

#### **What are the indications For Intensive Care Unit?**

- Severe dyspnoea that response inadequately to initial emergency therapy ,
- Change in the mental status
- Persistent or worsening hypoxemia (PaO<sub>2</sub> < 5.3kpa or 40mmHg) and /or severe /worsening respiratory acidosis (PH < 7.25) despite supplemental oxygen and non-invasive ventilation
- Needs for invasive mechanical ventilation
- Hemodynamic instability –needs for vasopressor

#### **What are the indications for Non-invasive Mechanical Ventilation (NIV)?**

- Respiratory acidosis (PaCO<sub>2</sub>  $\geq 6.0$ kPa or 45 mmHg and arterial P H  $\leq 7.35$ )
- Persistent hypoxemia despite supplemental oxygen therapy.
- Severe shortness of breath with clinical signs of respiratory distress like using accessory muscle of respiration, paradoxical motion of the abdomen etc
- Type 2 respiratory failure secondary to chest wall deformity, neuromuscular disorders and obstructive sleep apnoea.
- Cardiogenic pulmonary edema unresponsive to CPAP.



### What are the indications of Invasive Mechanical Ventilation?

- When patient cannot tolerate or failure of NIV
- Life-threatening hypoxemia in patients unable to tolerate NIV.
- Massive aspiration or persistent vomiting
- Severe hemodynamic instability without response to fluid and vasoactive drugs
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation.
- Severe ventricular or supraventricular arrhythmias.

### What is the criteria for discharge and recommendation for follow- up?

#### Discharge criteria

- Full review of all clinical and laboratory data.
- Check maintenance therapy and patient understanding
- Reassess the inhaler technique
- Ensure understanding of withdrawal of acute medications (steroids and/or antibiotics)
- Provide management plan for comorbidities.
- Ensure follow-up managements
  - Early follow-up <4 weeks and late follow-up 12 weeks as indicated while during each visit evaluate the patient thoroughly and address the concerns.

### COVID-19 and COPD

#### Mild COVID-19

- Continue COPD Therapy

#### Moderate COVID-19

- Systemic steroid, anticoagulation, remdesivir and IL-6 tocilizumab

#### Severe COVID-19/ARDS

- High flow oxygen, pruning, anticoagulation, NIV or Mechanical Ventilation

### Interventional & surgical therapies for COPD

1. Bullectomy
2. Endoscopic lung volume reduction surgery this may be unilateral or bilateral.
3. Endo bronchial one-way valves
4. Airways bypass stents.
5. Self-activating coils

Above all measures are mainly directed to reduce hyperinflation in emphysema.

#### Indication for lung transplant

- When Bullectomy and other intervention are not possible
- Pulmonary hypertension
- BODE index 7-10.



### Differences between Chronic Bronchitis and Emphysema

Features	Emphysema: (Pink Puffer)	Chronic Bronchitis: (Blue Bloater)
Diagnosis	Clinical	Pathological
Cyanosis	Absent	Prominent
Hyperinflation features	++	+
Dyspnoea	++	+
Cough	+	++
Cor pulmonale	+	++
Thin /obese	Thin	Obese
Pulmonary function test	TLC raised and DL co reduced. Static lung compliance increased	TLC normal and DLco normal. Static lung compliance normal
Ventilation-perfusion Testing	Increased ventilation to high V/Q areas i.e. high dead space ventilation.	increased perfusion to low V/Q areas

### Bullets points of COPD

#### Essential of diagnosis of COPD

- COPD is a major cause of chronic morbidity and is the third leading cause of death worldwide.
- One study of active smokers reported yearly decreases in FEV1 of 66mL per year in men and 54 mL per year in women compared to 30ml per year in men and 22mL per year in women who sustained smoking cessation. Therefore, smoking cessation is the single most important intervention.
- A 6 –minute walking distance of less than 350m is associated with increased mortality..
- Tran's nasal positive-pressure ventilation at home to rest the respiratory muscles is an approach to reduce dyspnoea in-patient with severe COPD.
- Increased exacerbations and mortality reported in some asthmatic patient treated with salmeterol have not been observed in COPD.
- CMDT 2023 labeled theophylline as fourth-line agent for treating COPD. Apart from bronchodilation, it has anti-inflammatory properties, strengthen diaphragm, and improve myocardial contractility and kidney function. However, it has narrow therapeutic window.
- Expectorant –mucolytic therapy is not helpful in chronic bronchitis.
- Apart from acute exacerbations, COPD is not generally responsive to oral steroid therapy.
- Supplemental oxygen for patients with resting hypoxemia ( $\text{PaO}_2 < 56 \text{ mmHg}$ ) is the only therapy with evidence of improvement in the natural history of COPD.
- Target O2 sat% in COPD patient and principal of O2 therapy in respiratory failure.
  - COPD patient with high  $\text{PCO}_2$  target O2 is 88-92%
  - COPD patient with normal  $\text{PCO}_2$  target O2 is 94-98%.



- type1 –high concentration (i.e. >35% usually 60%) /high flow (6-8L/min)
- Typ2 –low concentration (24-28) /low flow (1-2L/min).

### What are the Recent Advances in COPD?

Long acting anti-muscarinic drugs (LAMA) like Rovefenacin, Umeclidinium, and Acclidinium bromide.